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Catalytically and Noncatalytically Treated Automobile Exhaust: Biological Effects in Rats

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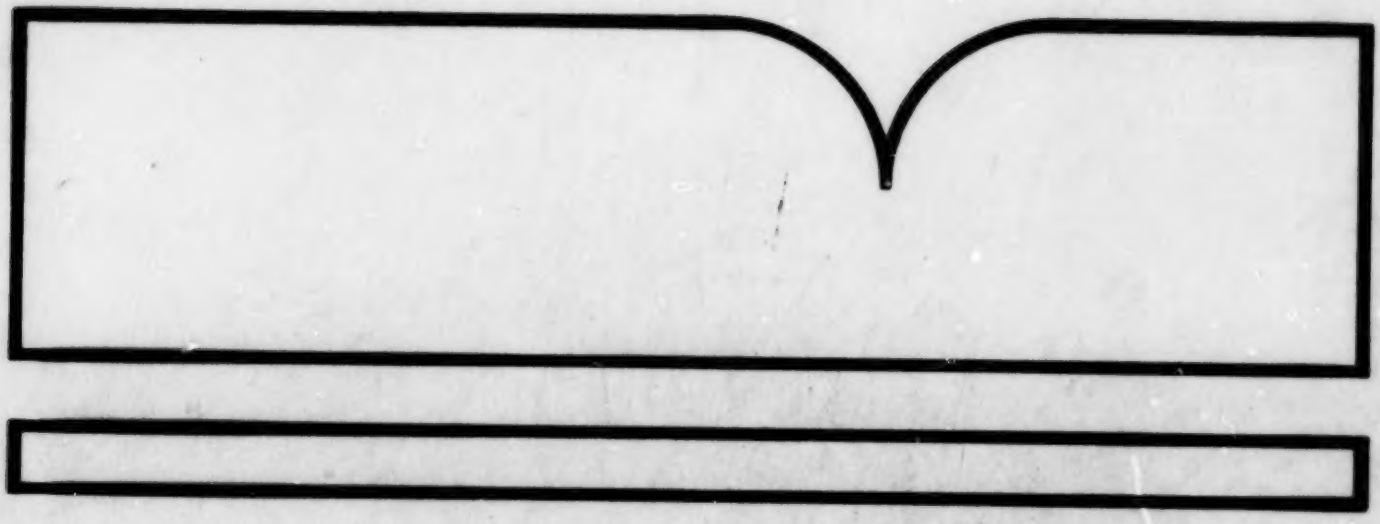

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CATALYTICALLY AND NONCATALYTICALLY TREATED AUTOMOBILE EXHAUST: BIOLOGICAL EFFECTS IN RATS

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Chronic exposure to catalytically treated or noncatalytically treated automobile exhaust significantly depressed the spontaneous locomotor activity (SLA) of rats. Exposure to H_2SO_4 alone or CO at comparable levels did not alter the SLA. Exposure to noncatalytically treated exhaust resulted in significant reductions in growth rate and food and water intake. However, these effects were not evident in the exposure to catalytically treated exhaust or in the control H_2SO_4 and CO exposures. Blood acid-base analyses indicated that exposure to either catalytically treated exhaust or H_2SO_4 elicits a metabolic alkalosis, while exposure to CO alone results in a metabolic acidosis. All acid-base parameters were within the normal range several weeks after the termination of exposure.

INTRODUCTION

The effects of automobile exhaust emissions on the quality of ambient air have been of concern for some time, and the biological effects of acute and chronic exposure to various levels of these exhaust components have

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been documented (Murphy, 1964; Hueter et al., 1966; Emik and Plata, 1969; Lewis et al., 1967). The installation of catalytic converters on new automobiles has resulted in a reduction of these pollutant emissions and a dramatic reduction of acute biological effects (Hysell et al., 1975; Lee et al., 1976).

However, there is still justifiable concern about the health effects of sulfuric acid (H_2SO_4) and other related sulfur compounds produced by automobile engines equipped with catalytic converters, since there is already evidence that these substances are toxic, particularly as measured by their effects on the lungs. Amdur (1969), in a review of the literature, pointed out that H_2SO_4 is a greater pulmonary irritant than sulfur dioxide (SO_2), the irritant effect being dependent on particle size. Furthermore, she indicated that sensitive individuals may show a reponse to levels of SO_2 as low as 1 ppm, an effect that is potentiated if particulates capable of oxidizing SO_2 to H_2SO_4 are present in the atmosphere. Others have found that chronic exposure to H_2SO_4 concentrations of 2.43 and 4.79 mg/m^3 produces changes in pulmonary function and structure in the cynomolgus monkey (Alarie et al., 1973).

Thus, there is a definite need to understand fully the effect of increased SO_2 and H_2SO_4 levels resulting from automotive emission control devices such as the catalytic converter. This study is one of the initial attempts to ascertain the health effects of emissions from catalyst-equipped systems as compared with those from engines not equipped with catalytic converters. An examination was made of the effects of emissions on the following group of general biological indicator tests: spontaneous locomotor activity, blood acid-base balance, food and water consumption, and growth. These exposures are part of a continuing research effort of the Environmental Protection Agency (EPA) for toxicological assessment of mobile emissions (TAME), begun in 1972.

METHODS

Exposure Conditions

A 1975 Ford 400 C.I.D. engine was used to generate the exhaust. The engine was run continuously, using the seven-mode modified "California cycle" (Hysell et al., 1975). The gasoline used was American Oil Company unleaded 91 octane Indolene motor fuel, supplemented with thiophene to produce a high-sulfur fuel (1,000 ppm). The average sulfur level in gasoline produced in the United States is approximately 400 ppm (EPA, 1976). However, a recent fuel survey indicated that the average sulfur level in leaded regular fuel in Los Angeles during the summer of 1976 was 660 ppm, but that the range extended to over 1,000 ppm (EPA, 1977).

In the TAME M study, the engine was run for 88 days using an Engelhard/American Lava substrate monolith catalyst in the exhaust pipe

system. TAME N used the same type of catalyst, but the engine was run for only 38 days. The TAME O exposure was the same length as TAME N, but in this case the catalyst was removed from the system.

Sulfuric acid control chambers were used in TAME M and TAME N in order to differentiate the effects caused by H_2SO_4 aerosol alone from the effects caused by the total exhaust emission containing H_2SO_4. A Retec X-70/N nebulizer was used to aerosolize an aqueous solution of the acid. The droplet size was reduced, before the aerosol was introduced into the exposure chamber, by passing the aerosol through a heated glass tube.

In TAME O, a reference carbon monoxide (CO) exposure chamber was used to distinguish between total exhaust effects and effects that could be attributed to CO alone. The CO used was obtained in cylinders from Union Carbide, Linde Division (CP grade). It was mixed with clean air in controlled proportions before introduction into the exposure chamber.

The exhaust emissions were diluted with approximately 11 parts of clean air and then passed through irradiation chambers prior to introduction into the exposure chambers (Table 1). In comparison, typical concentrations of some key atmospheric components on the downwind side of heavy freeway traffic were as follows: 6 ppm CO, 0.35 ppm nitrogen oxide (NO), 0.07 ppm nitrogen dioxide (NO_2), 0.005 ppm SO_2, and 0.140 mg/m^3 of total suspended particulate of which approximately 8% was sulfate (EPA, 1977). Essential features of the generation and exposure facility have been described elsewhere (Hinners et al., 1968). Test atmospheres were supplied to the exposure chambers at the rate of 15 air changes per hour, 24 hr/day.

Various precautions were taken to minimize any effect the animals might have on the composition of the test atmosphere. The insides of chamber walls were hosed with hot water daily. No more than ten animals were placed in each chamber and cage papers were changed daily.

The CO, nitrogen oxides (NO_x), and total hydrocarbons were monitored continuously, using automatic devices. The NO_2 was calculated as the difference between total NO_x and NO. The various exhaust components in the exposure chambers were measured by the following methods: CO, nondispersive infrared spectroscopy; hydrocarbons, flame ionization detection; NO_x, chemiluminescence; C_1–C_5 hydrocarbons, gas chromatography; C_6–C_{10} aromatics, gas chromatography; total aldehydes, colorimetry (Smith, 1972); SO_2, chemiluminescence and colorimetry (West and Gaeke, 1956); ammonia, colorimetry (Weatherburn, 1967); ozone, chemiluminescence. Particulates were characterized by the following methods: total mass, filtration gravimetry; size distribution, Andersen sampler; sulfate, colorimetry (Kufta, 1972); acidity, pH measurement. Both gas and particulate components were sampled several times a week. Determinations of the particulate size were made periodically. The size range of H_2SO_4 particles in the exhaust and in H_2SO_4 control chambers for both TAME M and TAME N was 0.20–0.30 μm mass median

diameter. Humidity and temperatures in the chambers were monitored daily.

Experimental Design

While each of the three exposures differed somewhat with respect to exhaust components and duration, the following procedure was employed in all exposures. In general, male weanling Sprague-Dawley rats were divided into wheel and nonwheel groups. Then each large group was subdivided into three equal subgroups. Of the three subgroups, one group was exposed to filtered air, the second to automobile exhaust, and the third to the reference atmosphere (either H_2SO_4 mist or CO). In the nonwheel group, rats in the three subgroups were housed individually (except two per cage in TAME M) for the duration of the experiment. Food and water intake and body weight were recorded weekly. The wheel group rats were housed individually in standard 36 cm running wheels. The wheel rats received exactly the same exposure as their corresponding group in the nonwheel condition. The number of wheel revolutions was recorded daily. Food and water intake and body weight were recorded weekly.

All animals were exposed continuously except for approximately 6 hr on 1 day/wk when cages were cleaned and food and water changed. After the rats had been exposed for the specified duration, all biological measures were monitored during a recovery period in which all animals were exposed to filtered air.

Near the end of the exposure period in all three TAME studies, blood samples were taken from the orbital sinus from approximately ten animals in the nonwheel group in each exposure. The blood was drawn into heparinized microhematocrit tubes, which were then sealed and stored in an ice bath. The blood was analyzed for pH, pCO_2, and hematocrit, and values for HCO_3^- and buffer base were estimated. All determinations were made by the same person on a London-Radiometer blood gas analyzer, and data were analyzed by using the *t*-test. Approximately 1 month after termination of exposure in all three TAME studies, another blood sample was taken from the same animals used during the exposure period and was analyzed as before.

RESULTS

Aerometry

TAME M. The aerometry data are summarized in Fig. 1, A and B. Each point plotted represents the mean value for all the data received from that particular week. It is evident that the hydrocarbon and NO levels were relatively constant throughout the study. The CO levels increased rather dramatically after the fourth week. This was the result of

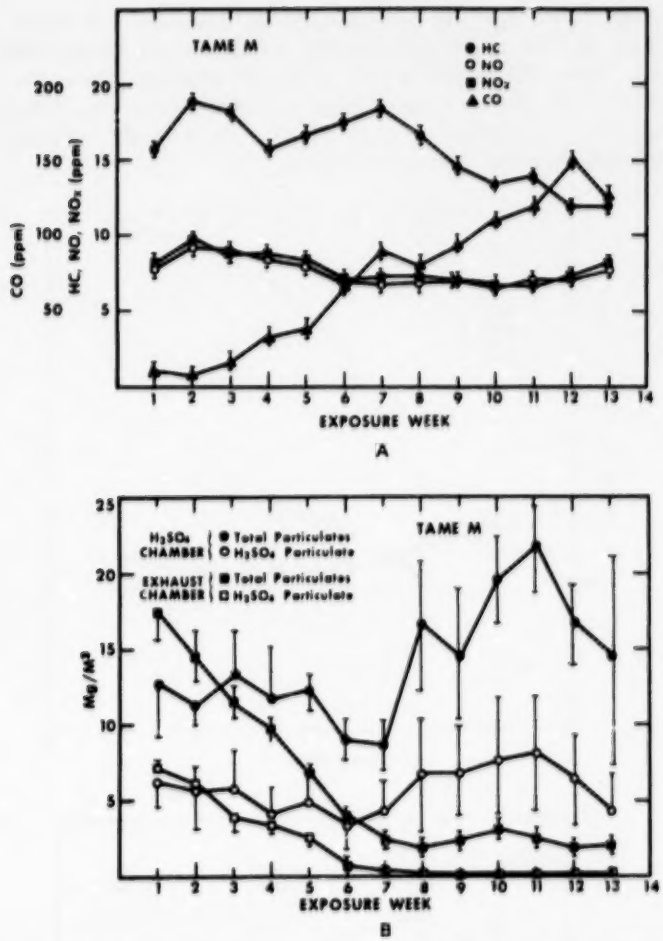

FIGURE 1. (A) Mean (± SEM) weekly concentration of total hydrocarbon, NO, NO_x, and CO in TAME M. (B) Mean (± SEM) weekly H_2SO_4 and particulate concentrations in TAME M.

changes in the properties of the catalytic converter caused by the process of aging under the conditions of these studies. The exhaust H_2SO_4 level decreased at approximately the same time. However, the H_2SO_4 level in the positive control chambers was not decreased; that is, no attempt was made in this experiment to duplicate the levels in the exhaust chambers. Instead, the levels were manipulated to ensure a mean H_2SO_4 level comparable to the level in the exhaust atmosphere at the beginning of the study.

TAME N. This exposure was shorter in duration and there was less variability in the CO and H_2SO_4 levels in the exhaust atmosphere (Fig. 2, A and B). The NO levels were relatively constant, but the CO and hydrocarbon levels did increase throughout the study. The exhaust chamber H_2SO_4 did not change; as a result, the levels in the exhaust and the H_2SO_4 control chamber were comparable.

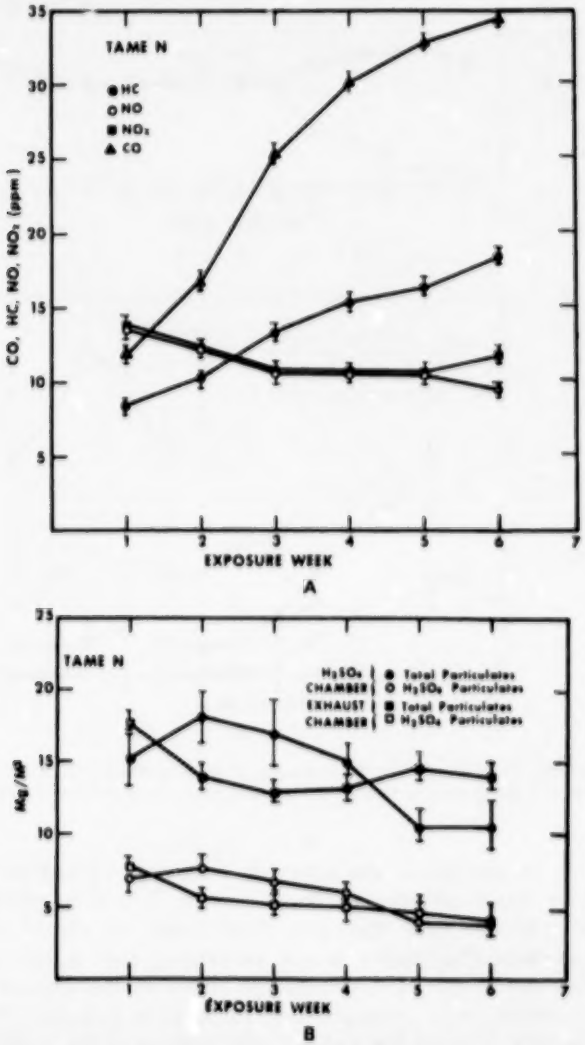

FIGURE 2. (A) Mean (± SEM) weekly concentrations of hydrocarbon, NO, NO_X, and CO during TAME N. (B) Mean (± SEM) weekly concentrations of particulates and H_2SO_4 during TAME N.

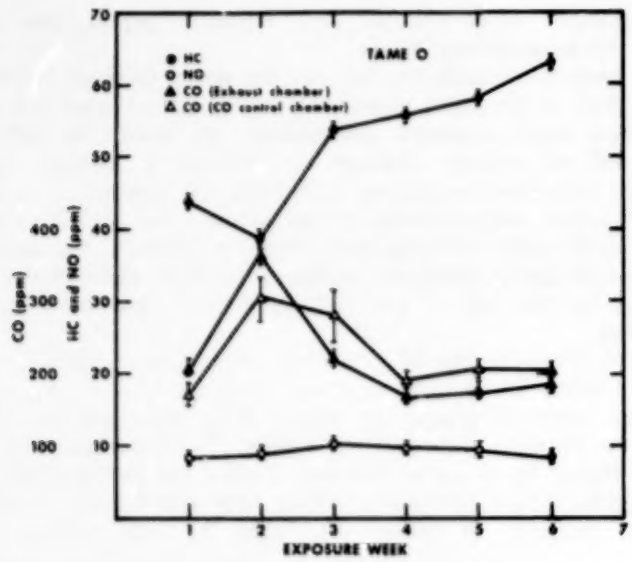

FIGURE 3. Mean (± SEM) weekly concentrations of hydrocarbons, NO, and CO in TAME O.

TAME O. The CO and hydrocarbon levels were much higher because no catalytic converter was used in this study. The level in the CO control chamber was comparable to the level observed in the exhaust chamber (Fig. 3). The NO levels were relatively constant.

The levels of various other types of constituents were measured in all three studies and are shown in Table 1.

Spontaneous Locomotor Activity, Food and Water Consumption, and Body Weight

TAME M. Twenty rats having a mean initial weight of 90 g were distributed randomly among three wheel groups, which were exposed to filtered air ($n = 7$), automobile exhaust treated with a catalytic converter ($n = 6$), or H_2SO_4 mist ($n = 7$). One nonwheel group of ten rats (mean weight, 95 g) was exposed to each of the three conditions. The results are summarized in Table 2. As shown in Table 2, no significant differences in either body weight or food and water consumption occurred as a result of the exposure. However, the mean activity level of the exhaust group but not that of the H_2SO_4 group was significantly depressed during exposure.

TAME N. In the wheel group, 24 rats (mean weight = 170 g at the beginning of the exposure period) were placed in three wheel groups with eight per group. As in TAME M, there were three exposure groups: filtered air, irradiated exhaust, and H_2SO_4 mist. In this experiment, the activity levels for all rats in the wheel group were monitored for 2 wk

before exposure. After the 38 day exposure period, the rats were monitored for an additional 9 wk.

In the nonwheel condition, ten rats per group (average weight, 149 g) were subjected to the same exposure conditions, but were not placed in the chambers until exposure commenced. As shown in Table 2, the activity level of animals exposed to automobile exhaust was again significantly depressed compared with both the control air and H_2SO_4 groups. All other measurements in the exhaust and sulfuric acid groups were not significantly different from those in controls. The activity level of the exhaust group remained depressed through much of the recovery period, but by the end it was not significantly less than that of the control group.

TAME O. Three groups of nine rats each (average weight, 110 g) were placed in running wheels and exposed to either filtered air, exhaust, or CO. In each nonwheel group, ten rats weighing approximately 108 g were subjected to the same exposure conditions. Exposure lasted for 38 days and was followed by 9 wk of recovery. Unlike the results of the previous two exposures, food and water intake was significantly depressed by automobile exhaust, as shown in Table 2. Body weight was also

TABLE 1. Exposure Atmosphere Component Concentrations in TAME Studies[a]

Pollutant	Atmosphere	TAME M	TAME N	TAME O
CO, ppm	Exhaust	70	25	210
	CO control	NA	NA	230
Total hydrocarbons, ppm, as methane	Exhaust	16	14	50
NO, ppm	Exhaust	7.1	10.8	8.3
NO_2, ppm	Exhaust	0.3	0.4	5.1
Hydrocarbons (gas chromatography), ppm				
Methane	Exhaust	3.23	4.07	3.85
Nonmethane aliphatics	Exhaust	1.56	1.33	4.41
Aldehydes, ppm	Exhaust	1.40	0.20	0.98
SO_2, ppm	Exhaust	2.22	1.31	2.42
Ammonia, ppm	Exhaust	0.044	0.033	0.016
Particulate, mg/m^3				
Total	H_2SO_4 control	14.85	15.18	NA (CO control)
	Exhaust	5.65	13.65	0.93
Sulfate	H_2SO_4 control	8.80	7.05	NA (CO control)
	Exhaust	3.06	5.91	0.14
H_2SO_4	H_2SO_4 control	6.59	6.35	NA (CO control)
	Exhaust	1.74	4.93	0.0

[a] The lengths, types, and exhaust dilution ratios of the studies were: TAME M, 88 days, catalytic, 11.6:1; TAME N, 38 days, catalytic, 10.9:1; and TAME O, 38 days, noncatalytic, 10.4:1.
[b] NA, not applicable; a different positive control chamber was used.

TABLE 2. Mean Daily Values for Spontaneous Locomotor Activity, Food Intake, and Water Intake

Measurement	Air control	Exhaust	H_2SO_4 or CO control
		TAME M	
Activity[a]			
Exposure	43.9 ± 5.3	27.5 ± 2.9[b]	38.8 ± 7.7
Recovery	26.6 ± 4.5	18.7 ± 2.3	29.0 ± 7.8
Body weight, g[c]	308.9 ± 18.0	293.1 ± 9.6	290.5 ± 6.8
Food, g	26.8 ± 1.3	26.2 ± 0.8	24.8 ± 1.1
Water, ml	57.2 ± 4.4	44.3 ± 3.8	49.9 ± 3.3
		TAME N	
Activity[a]			
Exposure	47.7 ± 4.1	25.7 ± 2.4[b]	49.5 ± 3.9
Recovery	41.2 ± 5.4	25.8 ± 3.1[b]	40.1 ± 3.4
Body weight, g[c]	344.4 ± 9.7	364.0 ± 6.2	356.6 ± 7.2
Food, g	25.7 ± 0.7	25.9 ± 0.5	27.2 ± 0.7
Water, ml	53.4 ± 1.9	49.2 ± 1.4	56.3 ± 2.4
		TAME O	
Activity[a]			
Exposure	27.6 ± 2.7	17.7 ± 1.3[b]	34.9 ± 4.1
Recovery	32.4 ± 5.4	39.8 ± 6.2	41.8 ± 9.2
Body weight, g[c]	215.4 ± 7.0	188.1 ± 7.0[b]	220.2 ± 6.4
Food, g	23.6 ± 0.8	19.2 ± 0.6[b]	24.7 ± 1.0
Water, ml	44.6 ± 2.4	35.0 ± 2.3[b]	49.9 ± 2.6

[a] (No. of revolutions)$^{1/4}$/24 hr.
[b] $p < 0.05$. Values are means ± standard errors.
[c] Body weight reflects average weight over the entire period analyzed.

significantly reduced. The mean daily running wheel activity of rats exposed to automobile exhaust was again significantly less than that of the control group. It is interesting to note that the CO group was actually somewhat more active than the control group, although this difference was not statistically significant. Thus, in this experiment, the depressant effect of other components of automobile exhaust on activity might actually have been partly offset by the effects of the CO contained in the exhaust. An analogous observation was made with respect to blood chemistry measurements. Activity levels and food and water intake in the exhaust group were comparable to those in the control group after only 2 wk of recovery, but body weights continued to remain depressed.

Blood Chemistry

TAME M. Table 3 shows the values for pH, pCO_2, HCO_3^-, and base excess (BE). In TAME M, one set of determinations was made near the

TABLE 3. Blood Chemistry Analyses

Measurement	Air control	Exhaust	H_2SO_4 or CO control
		TAME M	
pH	7.35 ± 0.04 (6)[a]	7.46 ± 0.01[b] (7)	7.47 ± 0.003[b] (7)
pCO_2	47.0 ± 2.47 (10)	54.3 ± 1.10[c] (8)	51.9 ± 2.17 (9)
Base excess, mEq/liter	0.2 ± 1.73 (9)	12.0 ± 0.99[b] (8)	11.4 ± 0.97[b] (9)
HCO_3^-, mEq/liter	26.2 ± 1.68 (9)	39.1 ± 1.16 (8)	38.3 ± 1.48 (9)
		TAME N	
pH	7.36 ± 0.02 (9)	7.41 ± 0.01[c] (10)	7.34 ± 0.02 (10)
pCO_2	52.1 ± 2.26 (9)	52.6 ± 2.22 (10)	59.2 ± 1.55[c] (9)
Base excess, mEq/liter	2.4 ± 1.26 (10)	6.9 ± 1.32[c] (10)	3.8 ± 1.32 (10)
HCO_3^-, mEq/liter	29.1 ± 1.36 (9)	33.5 ± 1.59 (10)	32.3 ± 1.44 (10)
		TAME O	
pH	7.38 ± 0.01 (10)	7.35 ± 0.02 (10)	7.32 ± 0.02 (10)
pCO_2	50.4 ± 2.49 (8)	50.7 ± 1.30 (10)	45.9 ± 2.30 (10)
Base excess, mEq/liter	4.1 ± 1.32 (8)	1.1 ± 1.15 (10)	−3.2 ± 0.98[b] (10)
HCO_3^-, mEq/liter	30.5 ± 1.61 (8)	28.0 ± 1.10 (10)	23.4 ± 0.75[b] (10)
Hematocrit	49.3 ± 1.64 (7)	52.9 ± 0.81 (9)	56.7 ± 0.93[b] (9)

[a] Number of observations.
[b] $p < 0.01$. Values are means ± standard errors.
[c] $p < 0.05$.

end of the 90 day exposure and the other was made 31 days after termination of exposure. During exposure, rats in the irradiated exhaust group and the H_2SO_4 group had significantly higher pH, BE, and HCO_3^-, indicating the existence of metabolic alkalosis. Thirty-one days after termination of the exposures, there was no difference between the groups.

TAME N. Once again, the HCO_3^- and BE levels were elevated in rats exposed in the H_2SO_4 chamber, but not enough to meet criteria for statistical significance. That these differences did reach significance in the TAME M H_2SO_4 control chamber might be attributed to either the longer duration of exposure or the slightly higher (compared with TAME N) acid levels—probably the latter. But the same explanation is not valid for the significant differences found in the exhaust chamber exposures in TAME M, since there was negligible acid present in those exhaust chambers during the final 6 wk of exposure. Once again, all measurements were not significantly different from control values 19 days after termination of exposure.

TAME O. In contrast with the H_2SO_4 exposures of TAME M and TAME N, CO exposure resulted in significant decreases in BE and HCO_3^- as well as the expected increase in hematocrit. Note that the same trends are present in the group of rats exposed to automobile exhaust, although

in no case did the changes reach criteria for statistical significance. Within 20 days after termination of exposure, all parameters were within normal (control group) limits.

DISCUSSION

It is clear that automobile exhaust, at concentrations used in these studies, whether treated with catalytic converters or not, depresses the spontaneous activity of rats. Previous studies of artificial smog (Boche and Quilligan, 1960) and polluted air (Emik and Plata, 1969) have demonstrated reductions of spontaneous locomotor activity of mice. It is also noteworthy that neither pure H_2SO_4 mist nor CO had such an effect; indeed, CO alone increased the activity level; thus, CO in the exhaust that was not catalytically treated probably counteracted a depressant action of the other exhaust components. It is thus apparent that the depressant effect of automobile exhaust in these cases must be attributable to components other than CO or H_2SO_4, or to the interaction of other exhaust components with H_2SO_4 or CO. Since the activity level was not suppressed by either H_2SO_4 or CO and since the level of NO_x was similar in the three experiments, it is reasonable to suppose that the suppression of activity results primarily from the hydrocarbon or nitrogen oxide components of exhaust. Other investigators (Gage et al., 1974), in studies of activity in mice exposed to automobile exhaust that was not catalytically treated, came to the conclusion that the hydrocarbon concentration was the principal factor responsible for the suppression of activity.

Animals exposed to H_2SO_4 alone or to irradiated automobile exhaust treated with catalytic converters show a shift in pH, HCO_3^-, and BE toward the alkaline side. These results were consistent throughout TAME M and TAME N. If it is assumed that acid sulfate produced such effects in exhaust chambers, it must be remembered that no measurable H_2SO_4 was present in TAME M during the last 6 wk of the 13 wk exposure period. Although urine acidity was not measured in these experiments, it may be reasoned that the alkaline shift could result from excess acid excretion by the kidney. Workers exposed to SO_2 have been shown to exhibit a higher incidence of increased urine acidity (Kehoe et al., 1932). Therefore, the alkaline shift in these experiments may result from the presence of SO_2 or other sulfates rather than H_2SO_4 itself. In TAME O the blood shifted to the acid side, significantly so in the CO chambers and less so in the exhaust chamber. This effect of CO was observed previously (Terzioglu and Emiroglu, 1957). In this study, the CO levels of the exhaust and CO control chambers were kept virtually identical, so that the less dramatic effects on blood chemistry of the automobile exhaust can be attributed to other components such as SO_2 or sulfates, which tended to offset the action of CO. To fully resolve the problems posed by the blood chemistry

data of these TAME studies, it may be necessary to examine the independent and interactive effects of hydrocarbons, NO_x, and sulfur compounds and to perform simultaneous renal function studies.

Finally, it is clear that the most dramatic overall effects were seen in animals exposed to automobile exhaust that had not been treated with catalytic converters. Only in TAME O were there significant changes in food and water consumption and body weight as well as in spontaneous activity.

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